BALTIMORE
MEDICAL COLLEGE

ALUMNI JOURNAL

PUBLISHED QUARTERLY.
Subscription Price, 50 cts. per annum.

VOL. I. APRIL, 1899. No. 4.

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Baltimore Medical College

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Vol. I. BALTIMORE, APRIL 1st, 1899. No. 4.

A STUDY OF METABOLISM IN TYPHOID FEVER.

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While much has been written in the last few years upon the subject of the treatment and diet of typhoid fever, the observations upon the relation of the food to the elimination by the intestine and kidney are scanty. A few observations have been published in this country during the past few years, but most of them refer only to the elimination of the various urinary constituents, no analyses having been made of the food or of the fæces. The sources of error in this respect are numerous and render any such experiments of comparatively little value. The quality of milk varies within wide limits and often it is removed from a common receptacle, with little or no care to obtain a uniform sample. Other liquids are often given which have even a more variable composition, such as egg-albumen, cocoa, etc. Another source of error arises when no analysis of the fæces is made. The amount of food actually digested and absorbed depends not only upon the functional activity of the stomach and intestinal canal, but also upon the peristalsis, patients with diarrhœa usually showing an increase in the amount of food products eliminated by the fæces.

Often also the analyses have been carried out by methods too inaccurate to render the results of any scientific value.

During a study of the urine of typhoid fever, the writer was struck by the high values of nitrogen found, as compared with those of patients suffering from non-febrile diseases, or from the irregular febrile disturbances of malaria and tuberculosis.

A preliminary study of the diet of typhoid patients showed that the actual amount of food ingested fell far below the values given for even the "subsistence diets" of the various writers upon metabolism, and led to the detailed studies given below.

The estimations of the food values were carried out by the following well-known methods: The proteids were determined indirectly by estimating the nitrogen according to Kjeldahl-Gunning, and multiplying the amount of nitrogen found by the factor 6.3. This method, while probably not strictly accurate, so far as the estimation of proteid goes, has the advantage of yielding values which may be compared directly with the amounts of nitrogen found in the urine and fæces. The carbohydrates were estimated by titration with Fehling's solution, employing the usual precautions to insure accuracy, viz: Dilution of the solution and boiling for two minutes, filtering and estimating the amount of copper in the filtrate. The fats of the food and of the fæces were determined by shaking out the substance, after rendering faintly alkaline with caustic potash, with petroleum ether, evaporating the extract at a moderate temperature, saponifying the residue with an excess of alcoholic caustic potash, and titrating the excess of caustic potash with a solution of sulphuric acid (semi-normal), using phenol-phthalein as an indicator. The milk was sampled by taking out each morning a sufficient amount for the succeeding twenty-four hours and placing in a separate receptacle, shaking well, pouring out the amounts necessary for chemical analysis, the whole being well mixed before the removal of each portion required for feeding and

measuring accurately the amount administered in a graduated cylinder. The same procedure was carried out for the other articles of diet.

The fæces were collected and prepared according to the method given by von Noorden, viz: Withholding all food during the night preceding the commencement of the week of the experiment and at about 3 A. M. giving a large dose of finely pulverized wood charcoal. The fæces were thrown away from the commencement of the week until they showed the presence of particles of charcoal, then carefully collected until the close of the week, when the same procedure—administration of charcoal, etc.—was repeated. The fæces were dried upon the water-bath, preventing the loss of any nitrogenous matter by the addition of a small amount of sulphuric acid, and finally drying to a constant weight in the hot-air bath at a temperature of 110 degrees C. The urine was collected as nearly as possible for each twenty-four hours and examined at once.

Case 1. E. H. entered the hospital August 4, 1897, after only about two or three days' illness. Personal and family history good.

The patient was a strongly built, muscular man, aged 30 years, weight about 180 pounds. There were two attacks of epistaxis in the early part of the disease, moderate bronchitis existed during the first week. The course of the disease was typical throughout; no complications, no hemorrhages, and only a slight tendency to diarrhœa. The Widal test gave a positive reaction on the eighth day. The fever dropped on September 2, never rising above 100 degrees F. after this date.

The treatment was the typical Brand; temperature taken every three hours; tub-bath at a temperature of 65 degrees F. whenever the rectal temperature rose above 102.6 degrees F.; diet exclusively liquid, consisting of milk, beef juice, egg-albumen and cocoa. Whisky was administered before and after each bath.

The following charts—1, 2, 3, 4—show the daily elimination by the urine, the amount of food administered, the amount of food material which escaped absorption by the intestine and the relation between the baths, average temperature, diet and elimination.

E. H.—CHART I.

Date.	Amount in cc.	Specific Gravity.	Reaction Acidity.	Albumen.	Indican.	Urea gms.	Uric Acid gms.	Total Nitrogen gms.	Chlorides as Nace.	Phosphates as P_2O_5	"A" H_2SO_4	"B" H_2SO_4
Aug. '97.												
11 to 12	1230	1020	4.990	....	Marked Excess.	32.59	0.9919	17.77	0.738	2.534	2.900	0.4029
12 to 13	1500	1015	4.536	....	"	33.75	0.8064	18.82	0.900	2.805	3.351	0.504
13 to 14	1400	1018	3.352	....	"	31.50	0.7256	18.58	1.120	2.646	3.116	0.3763
14 to 15	1275	1018	3.615	....	"	33.15	0.6854	17.49	Trace.	2.716	3.084	0.3427
15 to 16	2000	1014	3.150	....	Excess.	36.00	1.008	17.192	Trace.	3.32	2.839	1.2430
16 to 17	1100	1020	3.742	....	Marked Excess.	29.98	0.4435	17 93	Trace.	2.838	2.985	0.8316
17 to 18	1100	1022	3.257	....	"	27.50	0.6652	17.62	0.880	2.275	3.428	0.3604

E. H. CHART II.

Date.	Milk cc.	Egg albumen cc.	Cocoa cc.	Alcohol gm.	Proteid gm.	Nitrogen gm.	Carbohydrate gm.	Fats gm.	Calories.
August '97.									
11 to 12	668	170	0	13.2	26.67	4.234	45.39	18.33	558.
12 to 13	980	167	0	26.4	37.05	5.880	46.89	28.04	790.
13 to 14	720	149	85	26.4	34.22	5.432	48.50	27.36	778.
14 to 15	720	150	160	26.4	38.69	6.142	54.30	73.35	1248.
15 to 16	720	240	150	13.2	50.96	8.089	51.62	31.54	806.
16 to 17	720	248	90	0	45.36	7.201	52.76	28.36	666.
17 to 18	710	192	180	0	40.91	6.501	52.75	32.19	683.
Total,					273.85	43.479	352.21	239.17	5529.

E. H. CHART III.

Examination of Fæces.

Weight of faeces dried at a temperature of 110 degrees C..... 111.65 gm.
Nitrogen..... 3.04%=3.439 gm.
Fats .....38.55%=42.87 gm.
Carbohydrates.....Absent.
Percentage of loss in faeces :
Nitrogen in food..... 43.479 gm.
Nitrogen in faeces..... 3.439 gm.
Loss..... 7.9%

Fats in food..... 239.17 gm.
Fats in faeces..... 42.87 gm.
Loss..... 17.9%
Carbohydrates in food..... 352.21 gm.
Carbohydrates in faeces..... 0.
Loss..... 0.
Normal loss :
Nitrogen..... 6%
Fats..... 5 to 8%

E. H. CHART IV.

Date.	TEMPERATURE.			Baths.	Calories in Food.	Nitrogen in Food.	Nitrogen in Urine.	Nitrogen in Faeces.	Deficit in Nitrogen.
	Highest.	Lowest.	Average.						
August '97.									
11 to 12	103.2	101.6	102.7	3	558	4.234	17.77	0.491	14.027
12 to 13	103.0	100.6	102.2	2	750	5.880	18.82	0.491	13.431
13 to 14	103.0	101.2	102.4	2	778	5.432	18.58	0.491	13.639
14 to 15	103.0	101.4	102.5	2	1248	6.142	17.49	0.491	11.839
15 to 16	102.2	100.6	101.7	0	806	8.089	17.192	0.491	9.594
16 to 17	102.2	100.6	101.6	0	666	7.201	17.93	0.491	11.220
17 to 18	102.0	100.4	101.3	0	683	6.501	17.62	0.491	11.610
Total,								3.439	85.360
Daily Average,								0.491+	12.194

A study of Charts 1, 2, 3 and 4 show clearly that there is an enormous increase in the amount of nitrogen eliminated over the amount ingested, the daily deficit ranging from 9.549 gm. to 14.027 gm., with a daily average deficit of 12.194 gms., representing a daily loss of 76.82 gm. of proteid.

The digestion and absorption of the proteids, as shown by Chart 3, is fairly normal, the percentage of loss by the fæces being 7.9 per cent., instead of the normal, 6 per cent.

The digestion of fats is more affected, the loss by the fæces being 17.9 per cent., instead of the normal, 5 to 8 per cent. This high figure may, however, be due, not to an impaired digestion of fats, but to the small amount of fats in the food, the fats eliminated by the intestine normally being added to the amount not absorbed, thus apparently raising the percentage loss. Under a diet very poor in fats, it is a well-known fact that the fæces may contain more fats than are ingested.

The amount of the "B" or ethereal sulphates is much increased, varying, as shown by Chart 1, from 0.3427 gm. to 1.2430 gm., as compared with a normal elimination under a milk diet of from 0.120 to 0.200 gm.

Case 2. Mrs. V.. S., white, aged 36 years, widow, two children. Had the usual diseases of childhood, and an attack of acute articular rheumatism in 1893. The patient was a small, thin woman, weighing about 110 pounds.

The patient came to Baltimore from the country on August 26, 1897; had been feeling badly for several days previously, complaining of moderate diarrhœa, headache and loss of appetite. Ou August 30, the patient took to her bed and entered the hospital from her boarding-house September 5.

Blood examination showed a moderate degree of anæmia, absence of plasmodia, Widal reaction positive.

The case was one of moderate severity, running a fairly typical course except for a relapse which came on about ten days after the decline of the first attack.

The temperature charts for the period during which the metabolism was studied have unfortunately been mislaid, so that no comparison can be drawn between the temperature variations and the metabolism.

The treatment was the usual one of liquid diet (milk, egg-albumen and beef tea), cold baths at a temperature of 65 degrees F. whenever the rectal temperature reached 102.6 degrees F., the temperature being taken every three hours.

The study of the metabolism was commenced on September 6, the eighth day of the disease, counting from the day the patient took to her bed.

The following tables—5, 6, 7 and 8—show the urinary analyses, food analyses daily, the analysis of the fæces, and the relation of urinary nitrogen, food nitrogen and calories of the food : (*See Charts, pages 5 and 6.*)

The amount of nitrogen eliminated daily by the urine in Case 2 (Chart 5) varies greatly from day to day, which may be due to irregularities in the collection of the urine, at times a matter of difficulty, especially in women, in cases of mild delirium.

The figures when taken for the week as a whole, however, are fairly typical.

The amount of nitrogen ingested for the week is 42.571 gm., daily average 6.081 gm.; the amount eliminated by the urine 70.836 gm., daily average 10.119 gm.; the amount eliminated by the fæces 2.4064 gm., daily average 0.3437 gm; the total number of calories 4679, daily average 668. The amount of nitrogen eliminated for the week in excess of that contained in the food is 30.6709 gm., a daily average loss of 4.3815 gm.

The urine presented one very interesting feature in connection with the relation between indican and the ethereal ("B") sulphates. As will be seen from Chart 5, the indican was always excessive, and twice marked excess, while at the same time the ethereal ("B") sulphates show low values,

Mrs. V. S. CHART V.

Date.	Amount cc.	Specific Gravity.	Reaction.	Albumen.	Indican.	Urea.	Uric Acid.	Total Nitrogen.	Chlorides as Nace.	Phosphates as P_2O_5.	"A" H_2SO_4.	"B" H_2SO_4.
Sept. '97.												
6 to 7	750	1022	Acid. 2.4625	....	MARKED EXCESS.	19.875	1.4112	10.668	Trace.	0.9255	2.558	0.2016
7 to 8	290	1025	Acid. 0.877	....	EXCESS.	8.99	0.1169	5.440	0.166	0.4698	1.106	0.0974
8 to 9	700	1032	Alk.	Trace.	"	23.1	0.4704	13.328	0.700	0.5740	3.369	0.0294
9 to 10	550	1021	Alk.	...	"	14.575	0.3326	9.024	0.660	0.698	1.658	0.0924
10 to 11	290	1019	Faint Acid.	....	"	8.41	0.4640	4.466	0.464	0.4234	1.291	0.0414
11 to 12	1250	1020	Acid. 2.898	....	MARKED EXCESS.	30.76	0.9273	17.646	2.760	1.7590	3.226	0.1352
12 to 13	770	1019	Acid. 1.795	....	EXCESS.	17.33	0.5433	10.264	2.150	1.206	1.920	0.0840

Mrs. V. S. CHART VI.

DATE.	Milk cc.	Egg albumen cc.	Beef tea cc.	Alcohol gm.	Proteid gm.	Nitrogen gms.	Carbohydrates gm.	Fats gm.	Calories.
Sept., '97.									
6 to 7	720	150	0	6	38.90	6.174	46.90	29.52	668
7 to 8	720	269	0	12	45.48	7.220	49.87	25.60	713
8 to 9	540	160	60	18	33.13	5.258	31.26	26.16	633
9 to 10	540	174	6	12	31.33	4.973	31.16	19.37	521
10 to 11	540	190	60	18	38.38	6.091	33.53	22.56	631
11 to 12	760	110	60	18	39.47	6.265	48.16	29.68	760
12 to 13	720	130	120	18	41.61	6.590	46.55	28.49	753

Mrs. V. S.—CHART VII.

Weight of faeces dried at 110 degrees C. 75.2 gm.
Nitrogen ..... 3.2 % = 2.4064 gm.
Fats .....21.18% = 15.93 gm.
Carbohydrates.....Absent.

Percentage of loss in faeces:

Nitrogen in food .....45.57 gm.
Nitrogen in faeces ..... 2.4064 gm.
Loss 5.65%
Fats in food.....181.39 gm.
Fats in faeces..... 15.93 gm.
Loss ..... 8.78 gm.
Carbohydrates in food.....287.44 gm.
Carbohydrates in faeces..... 0.
Loss ..... 0.

Normal loss:

Nitrogen..... 6%
Fats..... 5 to 8%

Mrs. V. S.—CHART VIII.

DATE.	Calories.	Nitrogen in Food.	Nitrogen in Urine.	Nitrogen in Faeces.	Nitrogen Deficit.
September, '97					
6 to 7	668	6.174	10.668	0.3437	4.8377
7 to 8	713	7.220	5.440	0.3437	+1.4363
8 to 9	633	5.258	14.328	0.3437	8.4137
9 to 10	521	4.973	9.024	0.3437	4.3947
10 to 11	631	6.091	4.466	0 3437	+1.2813
11 to 12	760	6.265	17.646	0.3437	11.7247
12 to 13	753	6.590	10.264	0.3437	4.0177

much below those usually found under normal conditions. The urine when allowed to stand to undergo the ammoniacal fermentation, always assumed a bluish tinge and showed crystals of indigo upon microscopical examination; in addition, the urine showed a moderate reducing action upon alkaline copper solution. This was probably due to the presence of indoxyl in combination with glycuronic acid, instead of with sulphuric acid, as under normal conditions.

The digestive tract apparently suffered comparatively little injury in this case, the nitrogen of the fæces amounting to 5.65 per cent. of the nitrogen of the food, the normal being 6 per cent., the fats of the fæces 8.78 per cent. of the fats present in the food, against a normal loss of fats of from 5 to 8 per cent. Here, again, the slight increase in the amount of fats in the fæces may be explained by the small amount of fats in the diet, the normal elimination going on as usual, thus making the decrease in absorption more apparent than real.

The carbohydrates are perfectly digested, as would be expected from the fact that only carbohydrate easy of digestion and absorption (lactose) was taken.

In addition to the preceding cases, a few cases which have not been studied in such detail may be added to show that most of these cases have the same characteristics as those previously cited.

Case 3. L. M., colored, female, aged 38 years, very corpulent, initial weight about 200 pounds. Analysis made at beginning of the third week, diet and urine of August 5 to 6, 1897.

The diet consisted of milk, beef tea, and egg-albumen, containing 32.84 gm. of proteid, 5.196 gm. of nitrogen. The urine of the corresponding period showed the following characteristics:

Amount, 720cc.
Specific gravity, 1024.
Reaction, acid.
Albumin and globulin present, 0.247% (by weight), 1.798 gm.
Sugar, absent.
Indican, marked excess.
Urea, 21.6 gm.
Total nitrogen, 11.8944 gm.
Chlorides as Na. Cl. 1.44 gm.
Phospates as P_2O_5, 1.368 gm.
Sulphates as H_2So_4.

A + B	2.4735 gm.
A	2.1530 gm.
B	0.3205 gm.

Microscopical examination: Hyaline casts, cylindroids, renal epithelium.

In this case we find the amount of nitrogen in the urine (11.8944 gms.) exceeds that in the food (5.1960 gms.) by 6.6984 gm. No analysis of the fæces was made.

Case 4. E., white, male, aged 35; muscular man weighing about 185 pounds before the commencement of the disease. Analysis of urine made on the fifth day of the disease, the third day after his admission to the hospital :

Amount, 1100cc.
Specific gravity, 1026.
Reaction, acid.
Serum albumin and serum globulin present, 0.2109% (by weight), 2.3197 gm.
Sugar, absent.
Indican, marked excess.
Urea, 37.7 gm.
Uric acid, 0.9606 gm.
Total nitrogen, 20.6976 gm.
Chlorides as Na. Cl. 1.54 gm.
Phospates as P_2O_5, 2.849 gm.
Sulphates as H_2SO_4.

A + B	3.6589 gm.
A	3.3355 gm.
B	0.3234 gm.

Microscopical examination: Hyaline casts, cylindroids, renal epithelium and leucocytes.

From a study of the cases given above, we may draw the following conclusions:

1. The diet of the typhoid fever patient, as a rule at least, is insufficient in quantity and this insufficiency concerns all the food principles, proteids, fats and carbohydrates, consequently the caloric values.

2. No apparent connection can be traced between the variations in body temperature and the nitrogen elimination and loss (see Case 1, Chart 4).

3. That the amount of tissue waste, as shown clinically by the emaciation and demonstrated by the chemical analysis of the ingesta and excreta is probably not due entirely to the action of the protoplasmic poison of the typhoid bacillus, but in part to the long-continued under feeding.

4. That caloric values of from 500 to 800 are the rule in typhoid fever, instead of the 2,500 to 3,000 of the usual diet or the 1,800 calories of the "subsistence diet" of Playfair.

5. That the impairment of the digestive functions is not of such a marked degree as is generally supposed to exist, the absorption from the intestinal canal proceeding in a fairly normal manner when the small amount of food taken is considered. The normal figures given by von Noorden for the loss by the fæces of the various food constituents under a much more liberal diet being nitrogen 6 per cent., fats 5 to 8 per cent., carbohydrates 0, as compared with nitrogen 7.9 per cent., fats 17.9 per cent., carbohydrates 0 in Case 1; nitrogen 5.65 per cent., fats 8.78 per cent., carbohydrates 0, in Case 2. These figures must not be considered as exceeding the normal markedly when we consider the fact that the fæces must be considered partially as an excretion, the amount of which remains probably fairly constant, and when the amount of food administered is small, naturally tend to increase the apparent amount of food, which is not absorbed. As an example, we may cite the case in which, upon a diet practically free from fat, the fæces actually contained more fat than the food.

It may be argued that these cases have not received as much food as is ordinarily given to typhoid fever patients in hospital and private practice, and therefore do not represent a fair average. Every endeavor was made to induce them to take more food and with a liquid diet no more would be taken.

The text books, as a rule, do not enforce the point of sufficient feeding. Osler, in the last edition of his *Practice of Medicine*, states that three pints of milk is enough. Taking the average composition of milk as 3.15 per cent. proteid, 3 per cent. fats, and 4.5 per cent. carbohydrates, we find that the daily diet contains:

Proteid	47.25 gm.
Fats	45. gm.
Carbohydrates	67.5 gm.
Calories	888.9

As compared with the average normal diet containing:

Proteid	100 gm.
Fats	100 gm.
Carbohydrates	400 gm.
Calories	2980.

Tyson, in his text book of the *Practice of Medicine*, gives the amount necessary as

from four to eight ounces every two hours or from 1400 to 2800cc. In an article, "Urea-Estimations in Cases of Typhoid Fever Treated by the Brand Bath Method" (*American Journal Medical Sciences,* vol. 114, page 290), he gives a diet consisting of about 2000cc. of milk (72 ounces).

This would contain from the average figures for the composition of milk given above:

Proteid	63 gm.
Fats	60 gm.
Carbohydrates	90 gm.
Calories	1185.3

Great stress is always placed upon the fact that the diet should be entirely liquid, with the statement that it should consist almost exclusively of milk. The question may be asked, however: Is milk a liquid diet? It is, to be sure, liquid as it reaches the stomach, but (except in the rare cases of entire absence of the gastric juice) remains so only a short time, and then is precipitated as a hard curd of insoluble casein which must be fully digested before any absorption is possible.

The amount of carbohydrates and fats which have a marked influence upon the general digestibility of a meal and a distinct influence upon the body waste of nitrogenous materials are given only in comparatively small quantities. The influence of carbohydrates in limiting the amount of albuminous decomposition in the intestine may also be mentioned.

Albuminous decomposition, according to Neumeister (*Lehrbuch der Physiologischen Chemic,* p. 294), does not take place to any marked degree in culture media containing carbohydrates or in the intestine until the carbohydrates have been destroyed. It is possible that a part of the increase in the amount of intestinal albuminous decomposition may be due to the insufficiency in the amount of carbohydrates.

Experience only can teach how much food and how varied a diet can be given with safety in these cases, but it seems probable that the easily digested meats, properly cooked, eggs cooked or raw, gelatines, puddings of tapioca, well-cooked starch, etc., to which may be added butter, sugar, milk to suit the taste of the patient, can be given with perfect safety.

A more liberal diet will have much to do with the prevention of the post-typhoid anæmia, one cause for which is to be found in the very small amount of iron present in milk and egg-albumen and the usual typhoid diet. It will contain more iron in organic combination, practically according to the experiments of Bunge and his pupils the only form in which iron can be assimilated. We hope later to have an opportunity to study the effect of a diet based on these general lines upon the course of typhoid fever.

In conclusion, I wish to express my thanks to Professors Johnson, Streett and Warfield for their kindly co-operation in allowing me to carry out these observations upon their patients, and to my former assistant, Dr. E. B. Evans, for assistance in carrying out the analyses.

CALCIC WASTE IN PREGNANCY.

BY DR. W. A. MONTELL.

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Every dental practitioner has had brought to his attention the old adage, "For every child a tooth," and while attempting during his professional career to combat the fallacy of such a necessity has been forced to admit that during the period mentioned there has usually been a loss of the calcic material of the mother, which has been more or less evinced by the destruction of dental tissues and the impairment of fillings which we have viewed with pride.

In 1887 the writer was led to make some investigation looking to amelioration of these conditions, and submits the results, asking the members to draw their own de-

ductions from the facts submitted. Mrs. T., mother of two children, was under his care for dental services, and stated that she desired nothing permanent done, as in a few months she would be confined and would have to lose one or more teeth before the fulfillment of her time.

The writer combated the idea, and prescribed for her a tablespoonful of lime-water three times a day internally, and an alkaline wash of sodium bicarbonate and water twice a day. The child was safely born, and, like the others, a boy, but, unlike the others, whose temporary teeth were apparently frail, the last one, who has been with the others frequently under the writer's care, had dental organs of a character far superior to the first two, and the mother lost neither a tooth nor filling during the period of gestation.

Mrs. L., whose husband informed the writer that his wife was *enciente,* was given the same treatment, with similar result. She was the mother of four children previously, all of whom suffered from caries of their deciduous teeth, while the fifth was observed by the writer within the last year, and such teeth as were still retained showed no symptoms of caries, while those which had been removed by the usual physiological process were perfect except the usual absorption of the roots.

Mrs. A., to whose husband the writer suggested the treatment, used syrup of hypophosphites internally, and lime-water as a wash during pregnancy. She is the mother of two children, and shows the loss of dental structure herself, while the children, now four and six years old, have perfect teeth. There are nine more cases of similar results which the writer has carefully recorded, but the data obtained therefrom are not sufficiently complete to give in this paper.

While wishing to allow his professional brethren to draw their own conclusions, the writer wishes to record a few of his own views as deduced from the cases cited, viz: that undoubtedly there exists a good and sufficient ground to believe that during pregnancy there is an unusual strain upon the system of the mother for the construction and maintenance of the fetus, whose supply of nourishment is derived solely from the maternal source. That during this period the osseous structure of the mother being deprived of its usual pabulum, which is diverted for the purpose of the fetus, the secretions of the body and the condition of the blood are less alkaline than usual, and thus produces the chemical solution of the teeth; for the writer observed that the ravages of the structure of the teeth at this time appear to differ somewhat from the usual phenomena of caries, showing the effects of simple decalcification by acids on extracted teeth. And if the secretions of the various organs are, as we all well know, decidedly acid during this period, there must be a heavy strain and demand upon the system for such calcic constituents as are required during a normal condition; and the results of the writer's observation lead him to believe that we can supply this calcic material in a readily assimilated form through the use of lime-water and especially preparations containing organic compounds of calcium derived from the various cereals, not only preventing the usual loss of dental structure on the part of the mother and replenishing the strain upon her resources, but we can by this method give that foundation for the future dental organs of the fetus so much to be desired.

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*SELF-INFLICTED WOUNDS IN BOTH EYES, EARS, TONGUE AND LARYNX, LEADING TO THROMBOSIS OF THE SINUS TRANSVERSUS.

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BY GEORGE REULING, M. D.

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Professor of Ophthalmology and Otology, Baltimore Medical College.

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Mr. George P., 41 years of age, who for several weeks had been suffering from men-

*Read before the American Laryngological, Rhinological and Otological Society at Washington.

tal depression and religious hallucinations, by trade a painter, but recently occupied as a watchman in a Baltimore bank building, was on June 1 brought to the Eye and Ear department of the Maryland General Hospital in the following condition:

Sensorium greatly depressed, suggesting deep melancholia. Repeated questions would not elicit an answer save a dull groan; facial muscles, including frontal, paralyzed on right side, both eyelids on either side greatly swollen and bluish discolored, those of the right eye half open, those of the left firmly closed. On raising the upper lids by means of an elevateur the right eye showed a large hemorrhage in its anterior chamber, a small perforating wound of the sclerotic and the insertion of the rectus externus and considerable protrusion of the conjunctival transition fold, owing to intra-orbital hemorrhage. The eyeball itself was also slightly forced forward, amounting to 2‴, as measured by Keyser's exophthalmometer. Vision of this eye was reduced to only quantitative perception, but the visual field was intact.

The left eye was similarly injured, the wound of the sclerotic, however, being over the insertion of the rectus internus and extending slightly into the cornea, corpus ciliare and iris, a small prolapse of the ciliary portion protruding through the wound. The conjunctiva was suffused by large hemorrhages, which gave the appearance of an uninterrupted blood coagulum extending around the cornea and over the lower lid. The cornea showed all across its equator an abrasion of epithelium about 2‴ wide.

Vision of this eye was nil, evidently owing to the fact that the mechanical insult was sufficient to cause a total detachment of the retina.

Both ears showed already on superficial inspection several small abrasions of the cornea and auricles, as also some small subcutaneous blood spots and slight swelling of the parts. The meatus externus was filled completely with coagulated blood on either side. After a gentle use of the syringe and lukewarm, sterilized water, I could notice that the drum in each ear was torn by an irregular wound from which particles of tympanic tissue were hanging in shreds from the fissure. The lining membrane of the meatus externus showed also superficial scratches, but was not severely torn in any place. Hearing of the right ear: Loud voice in 4′, in left ear total deafness. Neither loud voice nor Rinne's test nor the perception by bone conduction (Wilber's test) have elicited any hearing. I must state here, however, that the deep melancholia of the patient and the difficulty to have him answer my questions made the acoustic examination, to my mind, somewhat doubtful.

The presence of blood coagulum in the middle ear did not permit me to determine the position, presence or absence of the ossicula.

On examination of the tongue a non-penetrating, stab wound was found in the centre, just at the beginning of the papillæ circumvallatæ and from the left tonsil downward a red streak of superficial abrasion extended toward the false left vocal chord, in which it evidently spent its force, as the true vocal chord was not injured.

All of the injuries described had been made with the end of a medium sized painters' brush handle of six inches length which the patient had trimmed to a sharp point with his pocket-knife. If we consider the systematic procedure of the man's actions, his deliberation based on the frenzied conviction that he would find forgiveness for his sins by depriving himself of those organs which he blamed for the commitment of his wrongs, we cannot but be astonished at how little he must have suffered while his frenzy lasted, during which each eye, each ear, his tongue and his larynx, were successively attacked. My treatment consisted of an iridectomy in the left eye for the purpose of removing the prolapse of iris and admitting of a closure of the sclero-corneal wound, followed by pressure; bandage over both eyes and the introduction of iodoform gauze

in both ears. In order to secure mental rest a large dose of chloral and bromate of potassium were given at 3 P. M., one hour after patient was brought to the hospital. Temperature was normal at that time. On the evening of the same day the temperature rose to 101½, patient tossed about in his bed and seemed disposed to tear the bandage from his eyes, so that morphine was used hypodermically and the patient guarded by a strong male attendant. The remainder of the first night passed off quietly and on the next morning I noticed an improvement in the acoustic of the right ear, which perceived common conversation in about four feet distance.

He also counted fingers with his right eye in four feet distance and seemed to be anxious to know whether he could recover. The left eye showed no improvement in vision, though the wound was beginning to close. The left ear was also completely void of acoustic perception, either qualitative or quantitative.

His tongue was greatly swollen and heavily coated; the left tonsil showed a false membrane of a rust brown hue and fetor ex ore of a slightly gangrenous character began to develop. After ordering a mouth-wash of thymoline, including a moist piece of lint over the tongue dipped in the same solution, I bandaged the eyes again and blocked the ears lightly as before. The sensorium of the patient was greatly improved at intervals, and his concern for getting well again and his regret for his acts became quite apparent. For the next three days the improvement of the right eye and ear were quite steady; fingers could be counted in fourteen feet on the fourth day and conversation voice be heard in ten feet. On the night of the fifth day the patient became delirious and of almost uncontrollable violence, so that two strong men had to hold him in check from one o'clock to six o'clock in the morning, when he fell into a profound coma, lasting thirty-six hours, when death, under muscular twitchings over almost every group of muscles of the body and a strong clonic contraction of the left sterno-cleido-mastoid occurred on the seventh day after the injury.

POST-MORTEM.

It was with the greatest difficulty that I was permitted by the widow and sister of the deceased to open the skull and inspect the brain. On removing the calvarium a considerable venous stasis over the whole convexity of the brain was seen, so that the meningeal veins appeared almost black and very tortuous. On digital pressure it was found that the left hemisphere was somewhat softer than the right one. After the opening of the dura mater and careful dissection, the left half of the sinus transversus was found to be occupied by a grayish red thrombus, which was tightly adhering to the somewhat greenish discolored walls of the sinus.

This discoloration was particularly noticeable in the fossa sigmoidea and along the sinus petrosus superior, a condition which was undoubtedly the result of a thrombophlebitis of the veins of the left inner ear which directly communicate with the sinus. The infection produced by the destructive stab wound which penetrated into the middle ear and most likely through the fenestra ovalis to the labyrinth has undoubtedly furnished in this case the seed for either an intra-otitic periostitis or an infectious thrombo-phlebitis, which led to the thrombosis of the sinus transversus and its near neighbor, the sinus petrosus superior. There were a few small capillary ruptures in the immediate surroundings of the thrombosed sinus, but no abscess or larger apoplectic deposit was found. The cerebellum and medulla oblongata were normal. As the family of the deceased would not permit the removal of the os-petrosum, I was obliged to reluctantly leave this interesting specimen in its original position untouched.

* *NEPHRITIS.*

THOMAS W. KEOWN, M. D. (95)

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Gentlemen—The symptoms of nephritis in its various forms are probably well known to you, and while I take the liberty of repeating some of them and making a few remarks I do so rather with the desire of provoking healthy discussion than with the thought that I am bringing anything new to your notice, nor do I presume on any originality in my remarks; but since the work in clinical laboratories has advanced so much of late and changed many of our old ideas, it has given us a wide field for research and tends to revolutionize the whole subject of nephritis, its etiology and line of treatment. It will alter many things that heretofore were marked by hard and fast rules. Dr. Bright first pointed out that albumenuria and dropsy were only symptoms of nephritis and not separate diseases. He first arranged the different forms of nephritis under their distinct heads and so clear and concise was the arrangement that after the lapse of more than half a century it still holds good in spite of our "rapid transit" methods. I will now briefly review some of the more important symptoms, beginning with the most common, albumenuria:

Albumenuria may almost to a certainty be considered as showing a temporary, if not in reality a permanent, lesion of the kidney, and, though some authorities (Senator, etc.,) regard albumenuria as a physiologic condition of all urine and have brought forward some very fine tests to support their view, yet we cannot quite agree with this extreme, and the difficulty with the tests is that they re-act with other substances, such as mucin, nucleo-albumen, etc., and it is not within the power of the average practitioner to distinguish between these substances and albumen when he uses these refined tests; hence we may say that the albumen which is not recognizable by the three tests most in use, viz: the heat test, Heller's test, and the potassium ferro-cyanide test, may be regarded as unimportant and left out altogether. Only when albumen is persistently present in the urine need any permanent lesion of the kidneys be considered, and a careful microscopic examination will be almost certain to reveal casts — epithelial, granular, and hyaline at different times. Albumen appears in the urine of some persons from time to time, in some after severe muscular exercise, in others after the ingestion of foods rich in albumen; again we find albumen in the urine of young people arriving at puberty. It is variable in amount and is not accompanied by any increased arterial tension, nor accentuation of the second sound of the heart; it generally disappears in the course of time. Albumen is also present in certain diseases with prolonged febrile symptoms or in a fever of short duration with high range of temperature, as in typhoid fever, diphtheria. Individuals who have shown albumen in the urine at any time of life often enjoy excellent health, and are never troubled with Bright's disease. Yet the kidney which permits the escape of albumen must be somewhat different from the normal which does not permit its escape; the injury may be trivial and nature repair it perfectly, but the longer the time needed for repair, or, in other words, during the escape of albumen, the more certain we are of this injury becoming a *locus minoris resistentiae,* and in due time albumen will begin to escape again. Some form of nephritis will make its appearance, with all its accompanying symptoms. It takes time, but the weak spot in the end gives way. The escape of albumen is said by some to be due to an altered blood state, by others that it is due to change

*Read before the Ptomaine Club, November 30, 1898.

in the histologic structures of the glomeruli; the latter is probably the better, but there are many able advocates on both sides and the burden of proof seems to hang in the balance. Dropsy is the most prominent symptom in the eyes of the laity—probably first attracts their attention by the swollen feet and puffed expression of the face—in some cases it is very much marked, especially in the acute and chronic parenchymatous forms. Wherever the swelling appears there is pitting on pressure, denoting the transudation of serum into the tissues; Bright supposed it was due to the loss of albumen making it more watery, thus allowing it to permeate the tissues, but in acute nephritis we have much dropsy and no loss of albumen to speak of; evidently this theory will not stand. Another view held was that there being little water excreted in dropsical cases, it was retained in the blood and found its way into the tissues, but in those cases where a calculus dams back the urine, even for days, we have no dropsy; hence it would be absurd to retain this view. If we take cognizance of the fact that dropsy occurs in acute and chronic parenchymatous nephritis and that the lesions in these two forms are chiefly glomerular in character, we may presume upon a very possible theory—the glomerulitis is caused by the excretion of toxic substances while passing through the fine vascular walls in the kidney structure. These toxic properties are not only in evidence in the blood vessels of the glomeruli, but also in the finer blood vessels throughout the system. The presumption, then, is that there are vascular changes elsewhere in the body which weaken the walls of the blood vessels and allows the escape of serum into the surrounding tissues; but sometimes we have great changes in the kidney structure and practically no œdema. This may be explained that the toxic power of the substances is greater, acting more quickly, on the one hand weakens the vascular walls generally, while on the other the excretion being kept up through a long period of time its greatest effect is shown at the point of excretion in the kidney. Dropsy is most marked in the acute and chronic parenchymatous forms which have large kidneys. It is not so much marked in the chronic interstitial form (contracted kidney). The power of the kidneys to excrete water bears some relation to the dropsy; for where the kidneys excrete water freely the dropsy is not marked. We have glomerulitis in the large kidneys most marked and dropsy in the tissues of the general system. It would be fair to presume, then, that the cause of the glomerulitis is also the cause of the dropsy.

The next symptom concerns the rate of excretion, especially delayed excretion of the normal constituents of the urine as urea, salts, etc., or in the extra work imposed by disease. The amount of urea excreted in cases of nephritis is generally far below normal, often taking two, three or four days to excrete the usual amount of one day's work (about 30 grammes). Now with the aid of a good laboratory the full analysis of a 24-hour specimen of urine may be found, but the average physician has neither the time, place, or ability to make such an analysis. In consequence some other test must be found to take the place of the total amount of urea excreted from day to day, which would be a fair guide for the rate of excretion. Methylene blue or potassium iodide may be used and noting the length of time their presence is found in the urine. One grain of methylene blue injected hypodermically will appear in less than 30 minutes and remains present in the normal urine for nearly 36 hours on an average. Now, if it should take two, three or four days to excrete all the methylene blue which ought normally to be excreted in 36 hours, it would mean that the kidney was unable to do work on time, would show a correspond-

ing delay in the excretion of urea and would point to an accumulation of urea or its derivatives in the blood and consequently a guide to a possible uræmic attack. Such incompetency on the part of kidney would doubtless be a valuable guide to the practitioner, enabling him to take such measures as he saw best adapted to the immediate welfare of the patient.

Bacteriuria, a symptom which is attracting much attention of late in laboratory work, promises to be one of great importance in the course and treatment of nephritis. Bacteria are found in the urine in many different varieties and in many diseases which have little to do with disease of the kidney structure, such as gonorrhœa and its sequelæ which may possibly reach the kidney along the genito-urinary tract. It will in time produce a nephritis, but we are concerned chiefly with those bacteria when they enter the urine through the kidney in the process of excretion. When, for instance, we find tubercle bacilli in the urine it is undoubtedly a sign of tuberculous ulcers along the genito-urinary tract somewhere and in many cases have been found in the kidney—in a few cases primarily so, more often secondarily. In typhoid fever we have the typhoid bacilli in the urine, and their toxines may produce the nephritis in the case; at least they can cause pyelitis; we have the presence of numerous streptococci and staphylococci in the urine in erysipelas, malignant endocarditis, scarlet fever, etc.; nephritis also accompanies these diseases.

A most important form of bacteriuria is that of the colon bacillus; patients with colon bacillus bacteriuria have usually associated with it intestinal disturbance of some kind. This bacillus can cause catarrhal inflammation and pyelitis; it may or may not cause the nephritis, but it is well worthy of consideration as a possible etiological factor. Habitual constipation, intestinal catarrh, carcinoma and typhoid fever are frequent associates of this form of bacteriuria (Roosing); our best authorities are of the opinion that nephritis is due to an infection, and some of the best examples of acute nephritis come from acute infectious diseases. Diphtheria and scarlet fever are good examples; in the former the infection is known, in the latter it has yet to be found out. Welch says that it makes very little difference in the character of the lesion whether it be due to the germ and its toxine or the toxine alone; it is much more troublesome to draw conclusions in cases of chronic nephritis as to cause than it is in the acute. Thayer has shown that chronic malaria produces in many cases a form of nephritis and scarlet fever is the most responsible cause for the contracted kidney, but the point to be made in this paper is the nephritis due to intestinal disorders of one kind or another, and the value of keeping the *prima via* open after the fashion of by-gone days. Woodbridge, in his treatment of typhoid fever, carries out this plan in addition to the possible disinfection, and the wonderful credit claimed for it must have its foundation in this point. He does not allow constipation to exist, nor tolerate the presence of germs there if he can avoid it. It is worth thinking about and acting on. The germ theory will soon find its way into the etiology of all diseases. We have but little groundwork on which to base our ideas of infection in nephritis, but some of it is very good, and the field is a big one; the harvest is ever ripe and the laborers are few, but the work will go on and soon we will have better results to report along this line; the relation between the intestine and kidney from an excretory point will have become disentangled, and whereas now we see through the glass darkly soon it shall be face to face.

(I am indebted to *The Philadelphia Medical Journal* and *International Clinics* for many points in this paper.)

THE VALUE OF THE PHARMACOPOEIA TO THE PHYSICIAN.

J. WALTER HODGES, M. D. (92)

The growing tendency on the part of physicians to regard the literature sent out by manufacturing houses is my excuse for presuming to call your attention to a work with which all are so familiar, but not so well acquainted. The pharmacopœia of the United States stands as the sheet anchor of medicine to the physicians of this country. Constructed as it is by the labors of bright men representing every medical and pharmaceutical college and organization of any note in the country it should call forth more appreciation than it is receiving in these later days.

The effort to obtain business through the doctors' indorsement of the various proprietary preparations with which the country is flooded has caused a great rivalry among the many houses engaged in manufacturing these medicines, and the manner of introducing them to the notice of the profession, by means of samples, circulars and agents taken from the ranks of the physicians, has made a great inroad on the old-time method, whereby the doctor thought for himself and constructed his own prescriptions. The specious arguments used by the manufacturers of the various patent and semi-proprietary medicines are so nicely framed that one is led into prescribing them unconscious of violating any principle of ethics. Just stop for a moment and glance at the great array of ready-made prescriptions prepared so temptingly. Have you a case of any kind that you are not asked to try one of the many gilt-edged, high-sounding panaceas on. The dictionary has been exhausted in furnishing names for them, so numerous have they become. The termination "ine" has been tacked on to every known disease in the universe, and every organ and tissue in the body to give you an instantaneously recognizable and infallible remedy with which to treat any trouble which flesh is heir to. Thus if you have a case of cerebritis, you may prescribe cerebrine; a cold in the head, give a prescription for blennostasine fevers, viz: phenacentine; for pain, phenalgine or exalgine. If your female patients do not respond readily to the desires of their husbands, you are asked to give them a little ovarine, or if the young men have not the courage of their fathers, some testine or penisine will surely fill the bill, and so on *ad nauseum*. It is not my intention in this paper to decry or belittle these preparations in any sense, for many of them are worthy of consideration. They are generally the result of some physician's labors along the line of therapeutic research, but my contention is that the doctor hurts himself in prescribing them, both morally and financially. For instance, a patient comes to you suffering from anæmia; you resolve in your mind the numerous drugs that experience has taught to be useful in such conditions, such as the tr. ferric chloride, ferric sulphate, oxide of manganese and other official preparations, but your memory suggests a preparation that has been frequently brought to your notice by the most nicely worded circulars containing testimonials of men of eminence, and scientific statements showing numbers of millions of red blood corpuscles produced by it in a given time, and you say to yourself "I will try this Gude's Peptomanganate of Iron on this case." You write a prescription for one bottle, to be taken as directed. The patient reads the prescription, remembers the name, takes the medicine and is possibly benefited, but who gets the credit? The doctor who prescribed it? No! no! Gude is given the praise, and the next time that patient or his friends are in a similar condition they go to a drug store and

ask for Gude's Pepto-manganate of Iron. The doctor has simply helped to advertise this proprietary preparation by which it is possibly sold to a dozen or more through his patient, and for which the doctor receives one fee, whereas he might by his knowledge of materia medica have constructed a prescription, composed of officinal drugs, and written in Latin, that would have brought him unbounded praise and more business.

This also applies to all such preparations as Sanmetto bromidia, Scott's emulsion, ung. resinol, tongalin, etc. It has been stated in the public press from time to time in recent years that while surgery has made rapid advances medicine has stood still. If this be true, I believe the cause in a great measure lies in the fact that physicians are too ready to give credence to the statements concerning compounds sent out by manufacturing houses and too little attention along the lines of research and experimentation with the standard drugs and chemicals described in the pharmacopœia. They certainly present a broad field for study. The pharmacopœia being revised every ten years, is allowed to embrace only those things that have been thoroughly tested and proved to be worthy of the physicians' confidence, discarding those that are of little or no value.

The doctor makes a profound impression on the mind of his patient when he gives him a prescription of his own making that relieves some grave malady, and inspires a confidence that will be evidenced by the great esteem and admiration with which the patient speaks of him to his friends. It is not necessary to enumerate the many drugs of high merit described in the pharmacopœia, but you have there described: Digestants, tonics, hypnotics, antispasmodics, stimulants, emetics, diuretics, expectorants, antiseptics, etc., awaiting your kind consideration and bidding you to try them, prove them and realize the reward their virtues will surely bring.

TUMORS OF THE BREAST.

A BRIEF HISTOLOGICAL STUDY.

BY CHARLES H. POTTER, M. D.

Bacteriology and Pathology, Baltimore Medical College.

Sixteen operations for removal of tumor of the breast during the past year in the clinics of the various surgeons of the Baltimore Medical College must be our apology, if one be needed, for writing on this well-worn subject, but among these tumors appear all the typical varieties which are customarily treated of in our text books and have been demonstrated to our present class in pathology because so typical.

So we shall hope to accomplish the double result of putting into more permanent form the work done by and for our present classes and also to keep our alumni, who by the hundreds have gone out from us to widely scattered fields, in closest sympathy with our present-day work and results from our pathological laboratory.

To simplify matters we throw out for the present two lipoma and also one very interesting case of comparative rarity—a genuine case of tuberculosis so closely simulating carcinoma as to need closest study under the microscope.

It is taken for granted that we today stand upon firm ground in regard to what constitutes a tumor and that all swellings of the breast are not true tumors, viz: swellings caused by suppurative processes as mastitis, simple cysts and swellings of gummata of syphilis or tuberculosis which we classify as infective granulomata.

These may be excluded in an attempted diagnosis of tumor by the examination of fluids drawn by aspirating needles in the two first instances, and the clinical history and yielding to treatment in the two latter.

Tuberculosis of the breast, if primary, would doubtless present considerable difficulty to diagnosis unless an area of casea-

tion should break down and show the bacillus of tuberculosis stained in the fluid.

The writer would also hope that the operation for removal of these tumors of the breast, at present looked upon as so simple in comparison with our brilliant laparotomies and hysterectomies, should take its proper place again in the minds of our younger surgeons, for no operation calls for more careful and thorough technique or scientific knowledge of surgery than this; for the radical operation for removal of tumor of the breast, if malignant, will take from two to four hours if the axillary and supraclavicular lymphaties are involved, such cases giving over 52 per cent. of permanent cures after a period of over three years, according to Dr. Halsted,[1] who gives a careful summary of nearly 150 cases in Johns Hopkins Hospital, and this certainly should be called brilliant surgery.

By " malignant tumors," which form over 80 per cent. of breast tumors (Billroth[5]), we mean sarcomata and many varieties arising from epiblastic cells, viz: squamous-celled carcinoma or epithelioma, adenoma, and carcinoma proper, the adenomata being today classified under malignant growths by advanced pathologists; one writer (Hansemann[2]) goes so far as to confess with sorrow that he made a report in one instance of "non-malignant fibro-adenoma," upon which the surgeon merely enucleated the tumor, but a few months later the tumor recurred as unmistakable carcinoma, from which the patient died.

The diagnosis of these malignant tumors before proceeding to operate will also be materially aided by carefully scrutinized family history, going back as far as facts relating to causes of death can be traced with certainty.

One remarkable instance of hereditary predisposition is given by Broca, quoted by Senn,[3] in which, beginning with the first generation, the mother died of cancer of breast in 1788; second generation, four married daughters all died of cancer, the oldest living to be 62 years of age; third generation, children of one of these mentioned above, one son and four daughters, died of cancer; another lost one daughter from cancer, and of this last mentioned all five children died from cancer, giving in one family history from 1788 to 1856 sixteen individuals who succumbed to this dread disease.

The differential diagnosis before operation between a scirrhus carcinoma and dense fibroma becomes very difficult in many cases, and it also often occurs that previously hard tumors break down, becoming gelatinous, so confusing the operator, and Ziegler[4] is right when he deprecates our old terms " carcinoma medullare," and " carcinoma scirrhosum."

But, leaving these general considerations, our readers will readily understand the peculiar interest to the pathologist, amounting almost to a fascination in tumors of this region, because of the manifold varieties possible from the two layers of embryonic cells from which all these tissues spring, viz: epiblast; from epidermis, squamous-celled carcinoma; infinite combinations of cells lining sudoriferous and sebaceous glands and hair follicles, giving many forms of carcinoma, with all the other possible combinations of carcinoma from lacteal glands and ducts.

From mesoblast, fibroma and myoma (several varieties of the former), lipoma, angioma, sarcoma (although rare) and possibly from deeper skeletal structures, chondroma and osteoma.

We present herewith briefly, from the pathological standpoint, two fibroma, one sarcoma and four carcinoma, omitting necessarily much which might be of interest from a surgical point of view.

Our technique is to harden thin blocks of tissue, taken immediately after the opera-

tion from various places in the tumor mass, in alcohol or Mueller's fluid, imbed in celloidin and stain in hæmatoxylin and eosin.

FIBROMA.

Case No. 762.—Breast, male, aged 19. Tumor mass under right nipple about 6 cm. in diameter and 2 cm. in thickness. Growth noticed about eighteen months before operation.

Microscopical: The fibroma cells are seen running in all directions around the ducts, taking such definite structure as to merit the name given by Ziegler,[6] "fibroma pericanaliculare."

The ducts are distorted by the bands of tissue and in some areas the gland cells are also much compressed and take the nuclear stain very deeply.

Very few blood vessels are visible, and some of the cells in the centre of the tumor are undergoing hyaline degeneration.

FIBROMA INTRACANALICULAR.

Case No. 352.—Breast, female, aged 32. Tumor of left breast, but extending over the median line to the right side, growing slowly till 18 cm. in breadth and 7 cm. in depth.

The tumor mass is dense and unyielding, and on cut surface the naked eye sees plainly the tortuous windings of the tumor cells, with minute clefts or fissures showing along longitudinally cut ducts.

Microscopical: The cells appear densely packed in bundles running in all directions in inextricable confusion.

Toward the border of the tumor a few glands and ducts slightly distorted, but in the centre all normal appearances have disappeared, and the ducts or canals are distorted into all conceivable shapes by the densely growing cells of the fibroma, giving the typical picture of intracanalicular fibroma, the tumor cells in early stages growing slowly into the lumen of the ducts, carrying the epithelial cells' lining before them, presenting themselves as polypoid fibrous ingrowths into the ducts, but in late stages completely filling the entire lumen and crowding the epithelial cells roughly into contact with each other.

These epithelial cells have many of them lost their cuboidal form and are huddled together so closely that nothing but the nuclei are visible.

SARCOMA ("ENDOTHELIOMA").

Case No. 1140.—Breast, female, aged 46 years, colored. Two small tumors of right breast—one about 5 cm. long and 3 cm. wide, the other about two-thirds as large, connected by narrow band.

Sections through tumor masses show glistening white fibres seeming to radiate toward the centre, with the appearance of a capsule surrounding the larger one.

Microscopical, with low power objective, the arrangement of cells closely resembles alveolar carcinoma, but the higher power reveals a lack of supporting connective tissue, and that the outer rows of cells making up the alveoli are in close contact with blood vessels.

The cells are somewhat spindle-shaped, possibly from pressure, and are arranged in rows concentrating toward the centre, like the spiral arrangement of the heart muscles in the vortex.

There is particular significance in the structure of the blood vessels running through the tumor cells proper, as all have poorly defined walls and always seemingly composed of a layer of cells resembling the tumor cells themselves.

In several places in the section under observation the vessel walls have dilated so much with blood as to simulate angioma.

There are no karyokinetic figures visible, even in the most actively growing parts of the tumor, such as one generally finds in large round-cell sarcoma and carcinoma.

CARCINOMA SIMPLEX.

Case No. 1069.—Breast, female, colored. Tumor mass below line of right nipple 12 cm. by 7 cm. and 3 cm. thick.

Skin freely movable and neither the nipple nor lacteal glands are involved.

Microscopical: Bebginning at the skin surface, it appears perfectly normal, with its underlying sudoriferous and sebaceous glands, with a fairly thick zone of subcutaneous fat.

Immediately underlying this, we come on a tangled mass of tumor cells, evidently from the glands of the skin. Some of them are arranged in definite sacculi, like the sebaceous glands, but considerably larger than the normal glands.

Another variety of groups of cells simulate clusters of sudoriferous gland, but without any ducts.

These groups of tumor cells are separated by a dense connective tissue stroma, and would probably give so firm a consistence as to be called scirrhus carcimona.

This variety of tumor is sometimes called adeno-carcinoma sebacium and carcinoma glandulare (Karg and Schmorl[7]).

ADENOCARCINOMA, COLLOID OR GELATINOUS.

Case No. 323.—Breast, female. Tumor 13 cm. by 9 cm. and 5 cm. thick, involving left breast, with ulceration about 4 cm. in diameter under nipple; skin adherent to tumor immediately surrounding ulcerated area.

Microscopical : Low power, beginning at the centre (gelatinous portion to be described below), there is an evident growth of tumor toward the skin, the cells being grouped like lobules of lacteal glands without ducts.

These are surrounded by dense fibrous bands of connective tissue with very few blood vessels, which probably accounts for the centre of the tumor breaking down.

The gelatinous portion consists of a framework of fibrous bands, which in the central area have also broken down, distended with the colloid material, the few remaining cells undergoing degeneration, a few showing droplets in the protoplasm of the cells.

A round-cell infiltration and evident new growth connective tissue would tempt one to call this case fibroma, in addition to the cancer proper.

This affords a practical illustration of a scirrhus breaking down and becoming medullary.

FIBRO ADENOCARCINOMA, CYSTIC.

Case No. 752.—Female, white. Tumor mass, 13x10 cm. and 4 cm. thick at centre; edges tapering off till almost wedge-shaped.

Many large cysts in central part of tumor nearest ducts, several measuring 3 cm. across the centres. These cysts filled with thin brownish-yellow liquid, which was lost on section.

Microscopical : Under the low power strong bands of fibrous tissue are seen surrounding the ducts and cysts in several places, also growing into the ducts, as in Case No. 352, described above.

The glands are also compressed by the connective tissue growth.

Evidences of active cell proliferation into surrounding tissues are unmistakable, although the glandular type of the cells has been much modified by the pressure.

ADENOCARCINOMA INTRA-CANALICULAR.

Case No. 646.—Tumor mass, 16x12 cm. and 7 cm. thick at its centre ; surface ulcerated and fungating.

Tumor was soft in parts, and a thin, pale yellow fluid could be squeezed out on pressure. On section there seem to be many degenerated areas containing the same fluid as exudes on pressure from the surface.

Microscopical : With the low power one would name this alveolar carcinoma, for everywhere, except at the very edges of the tumor there appear to be definite alveoli of connective tissue stroma lined by epithelial cells and the lumen filled loosely with the same type of cells not connected with the peripheral stroma.

High power. Many of these spaces have broken down ; some are filled with blood, thus differing from the tumors described by Dr. Halsted (loc. cit.), and there is also a very definite enlargement of blood vessels in the immediate vicinity, differing from the vessels in the sarcoma or endothelioma described above (Case No. 1140), in that a perfectly defined connective tissue framework surrounds these enlarged vessel walls.

The connective tissue bands separating the alveoli are gradually becoming infiltrated with cancer cells, and this is in some areas accompanied by a small, round cell infiltration, evidently new connective tissue.

The degenerations seem to be a variety of coagulation necrosis, with pigment granules taking the nuclear stain intensely, there is also hyaline degeneration of the walls of the arteries.

Although the central zone reveals an atypical arrangement of cells, as we go toward the surface we find the genuine adenocarcinoma grouping and a distinct intracanalicular growth in some places.

The individual cells are also far more uniform in size than in the older part, where varied sizes and also grotesque forms meet the eye. Many karyokinetic figures are visible.

There is not space to describe two other adenocarcinoma—one cystic, similar to those already described—and all of these described are among our common and typical tumors, with the exception of the last one.

The literature on the subject is so voluminous that we refer our readers to only a few volumes which may be readily accessible.

LITERATURE.

[1]A Clinical and Histological Study of Certain Adeno-Carcinomata of the Breast.—*W. L. Halstead (Transactions American Surgical Association, 1898).*

[2]Mikroskopische Diagnose der Boesartigen Geschwuelste.—*David Hansemann, Berlin, 1897.*

[3]Pathology and Surgical Treatment of Tumors.—*N. Senn, 1895.*

[4]General Pathology.—*Ernst Ziegler (Eighth Edition), 1895.*

[5]Krankheiten der Weibl. Brustdrüsen.—*Billroth, Stuttgart, 1886.*

[6]Lehrbuch Speciellen Patholog. Anatomie.—*Ernst Ziegler, 1895.*

[7]Atlas der Pathalogishen Gewebelehre.—*Karg and Schmorl, Leipzig, 1893.*

✣✣✣

A CASE OF RIGHT-SIDED HEMIPLEGIA ASSOCIATED WITH COMPLETE HEMIANAESTHESIA AND UNILATERAL MUSCULAR ATROPHY ON THE PARALYZED SIDE.

✣

BY ROBERT REULING, M. D. ('94)

✣

Former Assistant Resident at the Johns Hopkins Hospital.—Lecturer on Neurology at the Baltimore Medical College.

✣

Since Todd[1] in 1856 first called attention to the occurrence of muscular atrophy after intracranial lesions, the literature contains surprisingly few reports of cases descriptive of this condition. When Quincke[2] in 1893 reported seven cases from personal observation, he was able to collect but thirty-three additional cases published by other authors. The presence of a well-marked muscular atrophy appearing as it did in the case under my observation about three weeks after paralysis had occurred, warrants, I think, a report of the case. The sensory disturbances in this instance are also of special interest, as our knowledge of the sensory tracts in the brain has been so greatly enriched in the last few years that the presence of well-marked sensory disturbances is aiding in localizing more correctly pathological processes in this portion of the central nervous system.

REPORT OF CASE.

The patient, Sarah C., colored, aged 30; single; occupation, housemaid. Was admitted to the Maryland General Hospital on December 6, 1898. She was brought to the hospital in a semi-conscious condition, and an examination revealed a right-sided hemiplegia.

Family history unimportant, except that her mother had a "paralytic stroke" when 50 years old and remained paralyzed for the six years preceding her death. No history of a neurosis in the family.

Previous History.—Although never very strong and always of a small physique, her general health has been generally good. No history of rheumatism, pneumonia or typhoid fever. She denies the use of alcohol. No history of a primary lesion of syphilis or its secondary manifestations. Has never been of especially nervous temperament. No facts elicited pointing to the presence of hysterical stigmata.

Present Illness.—During the entire summer and fall of 1898 the patient enjoyed exceptionally good health, and was able to do the usual amount of work, etc. No history of frequent headache or attacks of vertigo. No dyspnœa noted. On December 6, 1898, while at work in the morning, she was suddenly seized with marked vertigo and a feeling of extreme weakness, affecting more especially the lower extremities. In attempting to reach a chair for support she fell to the floor, where her friends found her in a semi-conscious condition. She claims that at no time was she entirely unconscious, remembering much that transpired after her fall. After reaching the hospital her head ached considerably and this continued for several days. The vertigo was very noticeable for about two days, especially when the patient attempted to raise the head. There has been no vomiting either before or since the onset of paralysis. No convulsive movements noted in the extremities or face. During the afternoon of the day of admission a feeling of numbness became apparent; this gradually affected the right arm and lower extremity, so that she experienced the sensation as though these members had been separated from the trunk.

[1]Clinical lectures on paralysis, certain diseases of the brain and other affections of the nervous system. London, 1856.

[2]Uber cerebrale muskelatrophie, Zeitsch. f. Nervenheilkunde, Bd. 4.

At present she frequently has difficulty in locating this arm and leg in bed; when in the dark she uses the left hand to find them. At no time was there complete aphasia, though the speech was indistinct for several days after admission. No diminution in visual powers. No diplopia. Sensorium was slightly affected for the first week after admission, but she could generally be roused to answer simple questions satisfactorily. No difficulty in deglutition. No bladder or rectal symptoms.

Physical Examination.—I saw the patient for the first time on February 10, 1899, and found the following:

She is lying on her left side in bed. A very poorly nourished negro woman. Radial and temporal arteries show a moderate sclerosis. Pulse 105 to the minute, regular in force and rhythm, tension low. Temperature 98 (there had been fever at irregular intervals, with a maximum rise to 103). Chest expansion poor, but equal on both sides. Lungs clear. Heart-impulse felt in fifth left intercostal space under nipple, slight accentuation of aortic second sound, no murmurs. Examination of abdominal viscera negative.

Sensorium clear; patient answers questions intelligently. No sensory aphasia. The mouth is drawn to the left and its left angle slightly raised. The right nasolabial fold absent, the left exaggerated. All facial muscles supplied by the lower branches of the right facial nerve paralyzed. Frontalis and orbicularis appear normal. No ptosis present. The tongue when protruded deviates markedly to right; no atrophy of the organ. Pupils equal, react to light and accommodation. Visual fields normal. Ocular muscles show normal innervation. Hearing and taste normal.

A complete paralysis affects the right upper and lower extremities. In the upper this paralysis is of a flaccid character, the muscle tonus being absent. The lower extremity shows considerable rigidity (lead pipe contracture) and the leg is flexed on the thigh, the thigh towards the abdomen. What is especially striking is the advanced muscular atrophy in these extremities, this being especially true of the arm, none of the muscles being apparently spared. The biceps is only represented as a thin fibrous band. The right hand is slightly swollen (œdema), especially the dorsum. The following measurements show the differences in the two sides :

	Right upper extremity.	Left upper extremity.
Over mid. biceps region	15 c. m.	18 c. m.
5 c. m. above elbow joint	14 c. m.	17 c. m.
Midway between elbow and wrist	14.5 c. m.	15 c. m.
Greatest measurement of forearm	16 c. m.	19.5 c. m.

	Right lower extremity.	Left lower extremity.
Upper third of thigh	31 c. m.	34 c. m.
Middle third of thigh	29.5 c. m.	31 c. m.
Lower third of thigh	26 c. m.	26.5 c. m.
Middle leg region	21 c. m.	21.5 c. m.

None of the atrophic muscles show fibrillary tremors. Unfortunately an electrical examination was not possible at the time. Dr. Southwick, resident physician to the hospital, tells me that this atrophy of the muscles became apparent three weeks after the patient's admission.

The following sensory disturbances exist: There is a complete anæsthesia to touch, pain and temperature (heat and cold) and absence of the muscle and stereognostic sense (shape of objects, etc.) on the paralyzed side; this complete hemianæsthesia ends abruptly at the median body line. (See charts.) The mucous membrane on the right half of the mouth and palate also showed a diminution of sensibility. The conjunctiva of the lids on the right appeared less sensitive to painful impressions than that of the left side. I do not believe the cornea was anæsthetic. Since the studies of von Frey[3] and Nagel[4] have shown that areas

[3]Beitrage zue Physiologie des Schmerzsinnes-Bericht der Mathemat. Physik. Klasse der Kgl. Sachs Gesellsch. f. Wisensch. Leipzig, Juli, 1895.

[4]Die Sensibilitat der Conjunctiva u. Cornea des Menschlichen Auges. Pfluges Arch. f. die Ges. Physiologie, Bd, LIX, S. 563.

of anæsthesia exist on the normal conjunctiva and that a special instrument is required for such investigation, one can speak with little certainty on this subject.

As to the most likely pathological lesions which caused the paralysis, etc., in this case there can be little doubt, from the sudden nature of its onset, that either a hemorrhage occurred in the brain substance or that a sudden disturbance in the blood supply of certain areas from thrombosis or embolism of the cerebral arteries took place. The mildness of the apoplectic insult—the patient not losing consciousness and her general condition improving rapidly—would suggest that if a hemorrhage was the cause, it involved no very large surface. The absence of the usual causes (mitral disease, infectious processes, recent labor, etc.) for the formation of emboli tend to exclude this etilogical factor and it seems more likely that a thrombus had formed in a sclerotic cerebral vessel whose endothelium being injured produced a favorable site for such a process. It is just an onset with a milder apoplecti-form attack which is characteristic of sudden disturbances in the blood supply of the brain from such causes. Premonitory symptoms are frequently wanting. (von Monakow.)

Before taking up the sensory disturbances, I am fully aware of how careful we should be before attributing these to organic changes, involving sensory tracts, for it has been so frequently demonstrated (Charcot, Oppenheim, and recently van Oord[5]) that hysterical anæsthesia not infrequently complicates changes due to organic lesions. The presence of any stigmata of a functional neurosis must be carefully investigated. I believe such are fairly well excluded in this case.

In the lower portion of the medulla oblongata the sensory fibers of the posterior columns of the cord end in two large nuclei situated on either side of the raphe; the fibers of the column of Goll ending in the nucleus funiculi gracilis, while those of the column of Burdach end in the nucleus funiculi cuneati, the former nucleus receiving the sensory impressions from the lower extremity, while the last named receives those of the upper extremity. In close relationship with the cells of these nuclei are the endings of sensory neurons of a second order which convey these impulses to higher centres. These fibers cross to the opposite side of the medulla in their course to the mid-brain, this crossing of the fibers forming the lemniscus. Some of the fibers of the lemniscus end in the medulla, others pass to the pons and cerebullum. The main portion, however, passes upwards to end in the ventral nuclei of the optic thalamus; here neurons of a third order collect the sensory impulses and convey them to cells in the brain cortex (in the lower parietal region, perhaps in the central convolutions). It is clear that the sensory fibers in the brain are not unbroken paths, but are made up of segments (neurons) which increase in complexity as they near the cortex, and it is only in the more inner portions of the brain where these fibers are apparently collected into well-formed tracts; on nearing the cortex they diverge markedly so that their endings cover a large surface. The posterior third of the internal capsule (sub-thalmic portion) is made up principally of sensory fibers. All sensory impulses from the cord do not, of course, take the course described; some undoubtedly avoid the nuclei of the columns of Goll and Burdach and pass upwards in lateral tracts; this is undoubtedly true of the fibers subserving the sense of pain.

From the course and arrangement of the sensory fibers in the brain it is apparent that if a lesion is producing complete hemianæsthesia, this must be of considerable extent, provided it occurs in the cortical or even subcortical substance, for here the fibers have converged and take up a large surface, as before pointed out. That cortical lesions may produce hemianæsthesia

[5]Tabes ohne ataxia mit Hysterie. Deutsch. Zeitsch. f. Nervenheilkunde, Bd. 13, No. 1-2.

we see from the cases reported by Vetter, Nothnagel, Luciani, Gepilli and Starr and others.[6]. I believe that only a comparatively small surface was involved in the case here reported and that it probably has involved the sensory portion of the internal capsule of the left cerebral hemisphere. It is more difficult to exclude the sensory portion of the medulla, but there being no symptoms suggesting pressure on any of the cranial nerves. Of course, the facial was involved, but one could almost exclude the possibility of this being due to injury to the trunk of the nerve or its nucleus in the medulla, as the frontalis muscle was not paralyzed. Lesions in the lower portion of the medulla are generally associated with alternating anæsthesia (Starr[7]) or bilateral hemianæsthesia. In the latter instance one-half of the body is usually more anæsthetic than the other, these changes being due, of course, to the crossing of the sensory fibers in this portion of the medulla.

There is at present no very satisfactory explanation for the muscular atrophy following cerebral lesions. Charcot and his pupils, Pitres and Brissand believed it could be explained by the degenerations occurring in the pyramidal tracts and that this extended to and gave rise to secondary degenerative changes in the motor cells of the anterior horn and as these are also the trophic cells for the muscles, the muscular atrophy seemed easily explained. Senator in 1879, and later Baginsky, demonstrated that this muscular atrophy could occur without such changes in the anterior horn cells, so that the theory of the Charcot school no longer held good. Darkschewitch[8] holds that the muscular changes are secondary to changes in the joints (arthropathies), but as these are by no means constant, although comparatively frequent complications in these cases, his views can hardly be accepted. In my patient no true arthropathy existed, the increased size of the right hand being certainly due to a serious infiltration, probably a vasomotor phenomenon. Some observers still believe in the presence of special trophic nerves in the central nervous system and associate all such changes in the muscles, etc., to an interference in their functions, but as physiologists so far have been unable to demonstrate these satisfactorily, their existence is doubtful. The views of von Monakow, I believe, explain the muscular changes better than those of any other observer. He believes the atrophy is due to an absence or want of the several physiological impulses which are apparently necessary for the activity and growth of muscles; namely, the motor, sensory and vasomotor impulses which are constantly present; it is therefore the injury of several sets of fibers conveying these that produce these degenerative changes. It is certainly striking how frequently sensory and vasomotor phenomena are associated with the cases in which muscular atrophy appears soon after an intracranial lesion. That the muscles may atrophy within a remarkably short time after the onset of the paralysis has been demonstrated by Senator, who reports a case in which it appeared about the eleventh day; as a rule the atrophy begins between one and two months after the onset of paralysis. Cases have been reported in which the motor disturbance was very slight, so that a hemiparesis existed and marked muscular degeneration was present. After progressing for two to three months the atrophy usually remains latent and even perfect restitution of muscle substance has been reported. The muscles usually show slight diminution in their contractility to electric stimuli, and some authors have reported the presence of the reaction of muscle degeneration. (Eisenlohr.[9])

[6]See Von Manakow, Nothnagel's Spec. Pathol. and Therapie, ix Bd., S. 370.

[7]Medical Record, 1893, p. 168.

[8]Affection der Gelenke und Muskeln bei Cerebralen Arch. fur Psych, Bd. xxiv.

[9]Muskelatrophie und elektrische Erregbarkeits veranderungen bei Hirnherden. Neurol. Centralbl., 1890.

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Subscription Price, per year, 50c.

Address all communications to BALTIMORE MEDICAL COLLEGE, Alumni Journal, N. E. Cor. Linden Avenue and Madison Street, Baltimore, Md.

Vol. 1. APRIL 1st, 1899. No. 4.

WITH this issue of THE JOURNAL we round out our first year of publication. We wish to thank those whose ready support has helped to make our journal a success. Nothing but praise has come to us, and, while we know the many imperfections of THE JOURNAL, due largely to the inexperience of its editors and business manager, we still feel that THE JOURNAL is a creditable production, and this seems also the feeling of those alumni with whom we have been in correspondence. Our original intention was to start with sixteen pages of reading matter and eight pages of advertisement, but we had for our first issue twenty-four pages of reading matter and twelve pages of advertisements. Not less than twenty pages of reading matter has appeared in any issue. We are receiving letters from many of our alumni, inclosing subscription fees and writing encouragement. Many of them inclose lists of our alumni who are in their locality. We wish all of our alumni would do this. It is the only way in which we can secure a correct and complete list of our alumni and addresses. It is our desire to increase the number of issues next year to six, THE JOURNAL appearing every two months. In order to do this we ask the aid of every one of our members. Let every graduate of the Baltimore Medical College who wishes us success in our attempt to establish a creditable journal make that success sure by subscribing at once. We would like to start the second volume with at least five hundred subscribers, and we can do it if all who receive this JOURNAL will assist. We wish also to increase the amount of reading matter in each issue, and ask our subscribers to send us any interesting matter which they may accumulate in their practice. Let all our alumni remember that our interest in them has not failed, that their success will always cause us keen delight, and that a return visit to our walls will always be a pleasure to us, and that in their new field we follow them with our best wishes. We hope to retain a like regard and interest from them, and that the future of the school and the future of her graduates may each be enhanced by the glory of the other.

✣

THE next meeting of the Alumni Association will be held at the College Hall, Linden avenue and Madison street, on April 6 at 8 P. M. We hope all members, in the immediate vicinity at least, will find it convenient to be present. We have an interesting programme for the evening. Dr. J. W. Hartigan, professor of biology, University of West Virginia, will deliver the annual address. The alumni banquet will be held immediately after the close of the business meeting in the dental hall of the Howard street building. We wish a large attendance at both the meeting and banquet. The faculty and graduating class have been invited to participate and a good time is anticipated.

✣

ATHLETICS seem to have come to stay at the Baltimore Medical College. The new gymnasium is now an assured fact and we congratulate the future students of the College and the faculty on the matter. Our representatives in the Georgetown University games, competing with the best men from the University of Pennsylvania, Princeton, Georgetown and other schools, made good records, bringing home two silver cups as prizes in the running events, Mace, in one of the events, running second to Tewksbury of Pennsylvania, and beating Mulligan

of Georgetown. As these two men are at the top of the heap in racing matters, it shows that our men are able to hold their own in fast company. Our outlook for football is still more favorable. The indications now are that we will have the strongest team in the South. Altogether, our athletes and those not so athletic may look forward to a very pleasant and successful year.

COLLEGE NOTES.

BY DUNCAN M'CALMAN ('95).

"Go forth! the path of pain and hope is thine."

Our graduating class this year is smaller than it has been for very many years, owing to its members being the first to graduate under the four years' course. This has been the experience of medical schools all over the country, and was, of course, expected here.

Athletic activities have been wound up for the year, with the definite assurance of being renewed next year under more favorable conditions. Our gymnasium will by that time have been started under a competent physical instructor and in close proximity to the College. We hope for great things from this undertaking and have the right to expect a mental and moral, as well as a physical, improvement in the men who offset the laborious poring over books and lectures by the relaxation it will afford, and not only that, but our students will have in it a sort of home club in which they are interested, and to which they can at all times go, instead of wandering, as so many medical students in our large cities do, like lost sheep over the city, should they venture away from their boarding houses during their leisure moments.

C. W. Mace, Class of 1900, at Georgetown games, won the 50-yard handicap in 5 2-5 seconds, lacking but a foot of tieing the world's record. He also won the second place in the 50-yard scratch at the same tournament. At the B. A. C. games in this city he came out ahead in the 60-yard dash; time, 6 3-5 seconds. This equals the Maryland State record, and is but a fifth of a second behind the world's record. Mr. Mace is now looking for a trunk large enough to hold the silver pots and pans won by his fleetness of foot.

Our many graduates since '90 will be happy to learn of one of the interesting events among our little colony in Baltimore since our last issue, namely, the resignation of Dr. J. M. H. Rowland from the trials, tribulations and loneliness of a bachelor's life. Dr. Rowland, bachelor, is now no more! The minister signed the death certificate. Dr. Rowland, benedict, is flourishing. May our venerable (we have the right to so call him now) editor and Mrs. Rowland "live happy ever after."

The faculty is to be commended for giving scholarships to each year's class. These are obtained by competitive examination at the end of the session and entitle the winner to free tuition for the following year. Five such scholarships are now given, so that a boy, if successful, can enter and leave college after four years' time without having to pay any tuition. Prizes as such have been abolished, the student obtaining the highest mark receiving the usual gold medal and having *magna cum hande* prominently inscribed on his diploma, and the next six having *cum hande* similarly placed on their parchments.

Among those who took post-graduate courses with us during the session just closing have been: Dr. R. S. Keeler, Elderton, Pa.; Dr. J. W. McGuire, of Vondling, Pa.; Dr. F. H. DeVere, Dr. J. B. Peacock, of Cochran, Ga.; Dr. T. C. Blackburn, of

Boone, N. C.; Dr. L. A. Crowell, of Crimsic, N. C., and Dr. William Miner, from Nova Scotia. All of these, with the exception of Dr. DeVere, graduated here and did not feel as though they were strangers within our gates. Their post-graduate diplomas will, we hope, prove useful as well as ornamental.

The Alumni Association will be addressed this year by Professor Hartigan, of the University of West Virginia, at their annual meeting. This ancient order, with Dr. Rowland as its grand master for the present year, comes to life every twelvemonth. This year it demands a banquet in addition to its annual meeting, and a banquet it will have with the new-made alumni before the latter's paint is yet rubbed off.

Miss Morgan, the principal of the Training School for Nurses during the past two years, has accepted an important position in a preparatory school near Princeton, N. J., and, to the regret of her many friends in Baltimore, left for her new station a few weeks ago, Miss Pinyon, one of the school's graduates and former assistant, is filling her place with much acceptance.

Some students have the truly unfortunate idea that the money they pay as fees on entering a medical college is an equivalent in full for the teaching received. Did they but think for a few minutes, the fact would come home to them that the fees, even in the highest-priced institutions in the land, do not begin to compare with the outlay involved in the teaching of one man. This is self-evident without a thought additional as to the self-sacrifice of the teachers engaged. A word regarding the latter is timely: Take the Baltimore Medical College, for instance, and leaving the professors without mention, what real student who has given this matter a thought believes that the fees he paid are an equivalent for the patience, self-obliteration, earnest willingness to help and kindliness from day to day of such men as Drs. Brown in the Chemical Laboratory, Potter in the Pathological and Bacteriological Laboratories, Whitney in the Clinical Laboratory, Rowland in the Anatomical Laboratory, Marden in the Histological and Biological Laboratories, Bolgiano in the Operative Surgery Laboratory, with their assistants, the hospital and dispensary demonstrators? The pleasure of seeing an awakened interest, the grasping of an idea, a broadening of the mind in the student is a large part of the compensation these men receive, and with this they are content. Students ought surely to show their appreciation by doing good, solid work.

We wish the graduating class much success in their *debut*, and after; the first, second and third-year men much joy in their vacations, and very heartily a much needed rest to the Dean's office, Daniel included.

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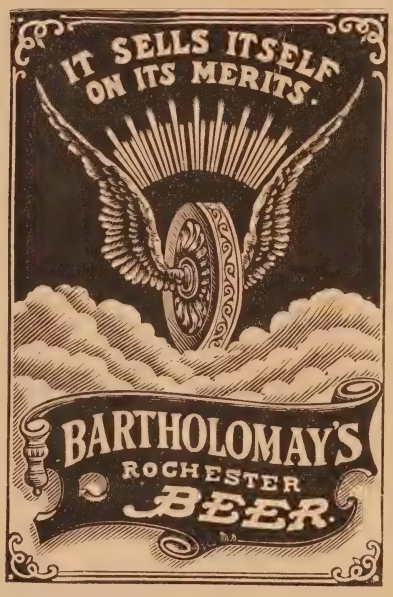

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